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Canine-Hysteria in Relation to Diet *†

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This paper has three aims, first to attempt in a brief manner to define the syndrome of dietary canine-hysteria; second, to present certain experimental studies which throw some light on the many factors involved in the aetiology of the disorder; and third, to attempt to point out how some of the results of these experimental studies may be related to clinical experience.

I The Syndrome of Canine Hysteria

The precise definition of the syndrome and of its differential diagnosis is a matter of considerable difficulty and one which may be left to another paper. Suffice to say that the term "canine-hysteria" is restricted to a completely reversible mental disorder in the dog, characterised by anxiety and apprehension, hallucinatory or confusional states of running and barking seizures, epileptic "grand-mal" convulsions and automatic behaviour, which has no organic morphological basis and which is not due to parasitic causes, be they viral, helminthic or bacterial, *i.e.*, the disorder is essentially a disturbance of the normal physiological functioning of the highest brain centres without organic disease.

Disorders of this kind have been recorded for many years. The 18th century compendiums on rural sports contain descriptions of various types of madness in dogs (Anon., 1735); some of course refer to rabies, but others might well be what we now term canine hysteria. In 1847 Elizabeth Barrett Browning gave an account (Huxley, 1931) of an illness in her dog, "Flush," which reads to-day like a typical attack of canine hysteria; it recovered in two days with the aid of castor oil, but both she and Robert Browning feared hydrophobia at the time.

During the latter half of the 19th century the disorder was described frequently under the term "epilepsy" (Moore, 1863) and it was not until about 1890 that the first references to hysteria appear, when Woodroffe Hill (1892) used the term in a vague manner to designate a syndrome of an epileptic character. It seems probable that the recognition of the disease as a separate entity coincided with the decline of rabies during the terminal decade of the last century. Since that time the term hysteria has become widely used in the British Isles, particularly since the mid-1920's when the condition became particularly prevalent. A similar disorder has been recognised as fright disease or "running fits" in the United States since the beginning of this century (Grace, 1930). On the continent of Europe, the disease has been virtually unknown in France apart from a series of short epidemics, which have been sharply localised geographically; a recent one swept the Riviera coast immediately after the landings of the American forces in 1944 (Saramito, 1946). In Holland the disease has been endemic since the mid-1920's (Klarenbeck, 1927). I have been unable to find any reference to its occurrence in Germany and Scandinavian countries or in Eastern Europe. In Australia the disease occurs sporadically (Parry, 1942).

Much has been written of the association clinically with certain foodstuffs, especially wheat (Hewetson, 1936; Arnold & Elvehjem, 1939; Parry, 1948) and the relation to certain batches of dog biscuits has frequently been stressed. It is the precise dietary factor involved that this paper will consider. Before discussing in more detail the role of wheat proteins, especially if they have been subjected to the agene process, it is necessary to point out that this syndrome occurs naturally under dietary regimens in which no wheat protein is being ingested; in this country outbreaks have occurred in kennels where in addition to wheat the cereal used was maize (Forsyth, 1947). In the Polar regions a similar syndrome known as Arctic or Eskimo Dog Disease occurs in dogs fed on meat alone (Baashuus-Jensen, 1935). There would thus appear to be a variety of aetiological types of dietary canine-hysteria, whose clinical manifestation is essentially similar. However, it is with the type of canine hysteria due to the ingestion of wheat proteins that the experimental studies have been carried out, and with which this paper will deal.

II The Experimental Disorder

1. The Experimental Method

The earlier experimental studies had employed various dog foods, which were found to produce hysteria spontaneously, but the occurrence of the hysterical episodes was extremely variable, and the conclusions of successive studies suggesting that the essential dietary defect was a vitamin A deficiency, a vitamin B_1 deficiency and a lysine deficiency have all proved to be incorrect. These studies, as well as many recent ones, are open to serious criticism in that no adequate precautions were taken to ensure that infective agents, intestinal helminthiasis and other factors known to produce epileptic states in dogs were not present.

In 1939 at the University of Wisconsin, U.S.A., the opportunity occurred of attempting to produce the disease experimentally by dietary means alone and with full safeguards to rule out infective agents. The dogs were obtained at weaning time from remote farms and housed in the strictest isolation. They were tended by myself only and no one else had access to them. They were treated with an anthelminthic and an ectoparasiticide and kept on wire-floored cages in a heated room, which was lighted artificially for about 12 hours each day.

It proved possible to produce the disorder in as short a period as 48 hours on certain diets adequate in all known nutritional factors required by the dog when given a normal healthful diet. The basal ration comprised commercial wheat gluten (which subsequent enquiry showed to have been prepared from wheat flour treated by the agene process) 487 parts, sucrose 633 parts, lard 377 parts, bone ash 40 parts, Wesson's salt mixture 30 parts and liver extract two per cent. by weight. To this was added once weekly, immediately before feeding, 10 mg. of α-tocopherol as the acetate. Two mls. of cod-liver oil, containing 1,500 U.S.P. units of vitamin A and 100 U.S.P. units of vitamin D, were given daily orally by pipette. Thiamine and pyridoxine hydrochlorides were given twice a week by pipette orally at a rate of 160 g. of thiamine base per 100 g. of food consumed and 100 g. of pyridoxine base per kg. body weight per day. From other studies it was known that the other members of the B-complex were adequately supplied by the liver extract. Precise daily records of individual food consumptions and body weights were kept, and in all 16 animals from three mongrel litters varying in age from eight to 48 weeks were used, some repeatedly after a short period of recuperation on a ration similar to that given above, except that the wheat gluten was replaced by casein.

Some 180 hysterical seizures were observed, and on the basis of these observations it is possible to describe with some accuracy the clinical signs of the dietary type of the disorder, uncomplicated by the many other factors which occur in natural cases.

2. Clinical Signs of the Experimental Disease

It is convenient to describe the clinical signs of the disorder in seven distinct stages, which may be divided into three main stages thus:

A. *Pre-hysterical State* of Hysteria-proneness.
   - I. Prodromal Anxiety Stage.

B. *Hysterical State* of (a) Psychomotor Seizures.
   - II. Barking Seizures
   - III. Running Seizures (Often combined as Running Barking "fits") and (b) Grand-Mal Epilepsy
   - IV. Epileptiform Convulsions.

C. *Post-hysterical State* of Recovery.
   - V. Automatic Behaviour
   - VI. Immediate Recovery
   - VII. Residual Defects.

I. *The Prodromal Anxiety*

The animals appeared extremely apprehensive, as if frightened of some imaginary situation or being. They were restless, did not

* The experimental observations upon which this paper is based were carried out at the University of Wisconsin during the tenure of a Commonwealth Fund Fellowship, 1938 to 1940.

† The substance of this paper was given at a meeting of the Mid-West Division at Bristol on Aprjl 16th, 1948.

play with their mates as usual and often hid in the darkest and most inaccessible part of the room. Other animals would come to the author as if seeking reassurance and would stay by the chair for long periods. In the severer phases the dog's tail would be carried between its legs, the ears laid back on the neck and the animal be taut and continually glance backwards over either shoulder. In very severe cases the animals would not respond to being called by name, which normally they would do readily.

II. *Barking Seizures*

The animal would bark continuously in a high pitched tone or with a low-toned howl, usually while standing quietly or sitting on its haunches. During the barking seizure the animal took little or no notice of its surroundings and would not respond to its name, nor would it heed admonitions to be quiet. On occasion these seizures occurred alone, but more usually they were combined with running seizures in the form of running barking "fits."

III. *Running Seizures*

The animal ran round and round its cage at a mad gallop making frantic efforts to jump out over the corners, although on occasions when the cage door was open no attempt was made to pass through the opening. The limb movements and gait were perfectly co-ordinated, the pupils dilated and the heart rate considerably accelerated. Although the animals would run into small obstructions such as a table or chair leg, they were never seen to run into a solid obstacle such as a wall, invariably turning from it and proceeding to the corner. Severe episodes lasted for upwards of five minutes and the animals would foam at the mouth; they would not respond to their name and if approached roughly would bare their teeth and snap at an approaching hand. However, if handled quietly, they were invariably docile and the duration and severity of these motor seizures could be reduced remarkably by sympathetic handling. In many instances the end of a seizure would find an animal cringing in the darkest and remotest corner. Although running seizures occasionally occurred alone, they were more usually combined with barking.

IV. *Epileptic "Grand-Mal" Convulsions*

These exhibited the three classical phases of "grand-mal" epilepsy, namely a premonitory phase followed by tonic and clonic phases. The premonitory phase was often very short and inconspicuous, but if noted carefully, a dog would be seen to become rigid, look straight ahead and occasionally show small bilateral symmetrical champing movements of the jaws. This was immediately followed by the tonic phase in which the animal became completely rigid with limbs extended and head and neck retracted, respiration ceased and the animal fell over on its side. After 10 to 15 seconds the clonic phase supervened with running movements of the limbs, while still lying on the ground, and respiration recommenced. The complete convulsion lasted in all 30 to 60 seconds. Consciousness was apparently lost completely; there was seemingly no recollection of what happened and there was almost always involuntary evacuation of urine and faeces, with drooling of saliva from the mouth. After a convulsion the animal would usually lie quietly for one to two minutes, breathing rapidly, as full consciousness returned.

V. *Post-dromal Automatism*

This stage normally occurred after either an epileptiform, convulsion or running seizure. The animal would appear apparently normal on casual inspection. However, it would keep aloof from its fellows, not respond to its name, and in several instances walk with an exaggerated raising of its fore-legs, and its body held rather stiffly, with the eyes fixed straight ahead. On several occasions dogs in this stage have walked through the open doors of their cages and fallen a couple of feet to the ground (without making any attempt to save themselves) where they have picked themselves up and walked on unconcernedly.

VI. *"Immediate" Recovery Stage*

If the hysterical episodes were prolonged the dog, especially if young, showed for some minutes unsteadiness on its feet and in very severe cases would lie exhausted for 30 minutes or so. In such cases, if the diet were not changed, death would ensue preceded by collapse and coma.

VII. *Residual Neurological Defects*

These occurred only in the younger animals which had been allowed a large amount of the hysteriagenic ration. They took the form of a dyssynergic ataxia lasting 24 to 48 hours, with defects of vision. Amblyopia persisted in several instances for some days; ophthalmoscopic examination revealed nothing of significance to account for this.

3. The Sequence and Frequency of the Clinical Stages

The most common stage was that of the prodromal anxiety from which the acute motor episodes usually followed, but not infrequently these motor episodes occurred without marked prodromal anxiety. The characteristic stage is, of course, that of the motor episodes, and the running barking seizure was the one most commonly observed, usually alone, less commonly either preceded or followed by an epileptic "grand-mal" convulsion. Automatic behaviour was an inconstant observation and usually followed an epileptiform convulsion, although very occasionally it apparently occurred as the only stage of an hysterical episode.

4. The Nature of the Dietary Defect

With a diet of purified constituents such as those employed it is possible to rule out certain possibilities regarding the dietary defect. Thus it is definitely not a nutritional deficiency of any known nutrient, certainly not any of the known B vitamins nor of vitamin A. The dogs were in nitrogenous equilibrium, and hence an amino-acid deficiency, say of lysine or of tryptophane, both of which have been incriminated, can hardly have occurred.

All the evidence points to the presence of a positive noxious chemical agent (*i.e.*, it is not a matter of the physical texture of the gluten) which is present in wheat gluten (certain samples particularly) and in gliadin prepared by the extraction of wheat gluten with 70 per cent. alcohol (Melnick & Cowgill, 1937). It is stable to heating to 400° F. in dog biscuits, in which it remains potent for years. This agent may be called a primary dietary hysteriagenic agent, *i.e.*, it has the power to induce in dogs a state of hysteria-proneness, from which motor episodes may occur spontaneously or be elicited by various factors. The quantitative relations between the primary dietary hysteriagenic agent and the onset of hysteria are of some interest. There is a very definite *maximum tolerated daily dose* (the M.T.D.D.) which an animal can ingest indefinitely without clinical signs of hysteria. Under my experimental conditions the M.T.D.D. was about 5 gm. of wheat gluten per kg. body weight per day. The animals will ingest amounts below this indefinitely without exhibiting any clinical signs of the disorder. At this level of 5 gm. per kg. animals would show only an occasional motor seizure, which was usually brought on by additional precipitating factors, of which mention will be made below. If the ingestion of wheat gluten is increased above the M.T.D.D., the number, frequency and severity of the seizures increase, until with levels above 20 gm. per kg. the animals show an almost continuous series of motor seizures, which appear apparently spontaneously. It is thus possible to get a wide variation in the occurrence of motor episodes depending on the amount of the primary hysteriagenic agent consumed.

Recently more information has come to hand as to the nature of this substance. Mellanby (1946; 1947) provided a valuable clue by showing that treatment of wheat flour with nitrogen trichloride in the "agene" process might be responsible for hysteriagenic potency. Moran and his colleagues (Moran, 1947; Bentley *et al.*, 1948) have confirmed this suggestion and have shown that primary dietary hysteriagenic agents can be produced in a wide variety of proteins, *e.g.*, casein, zein of maize, in addition to wheat flour, if they are exposed to nitrogen trichloride, especially if the concentration of the gas be raised far above the levels normally used in milling practice. They suggest that the toxic agent may be derived from the methionine portion of the protein molecule. It seems likely that before long workers in the U.S.A. and in this country will have isolated the agent in a pure chemical form. When this has been done it will be possible to examine the pharmacological properties of the substance more fully; the substance seems to be particularly active in the dog, and possibly in the cat, as other laboratory animals such as rats, mice, guinea-pigs and monkeys are not affected clinically, although monkeys show small changes in cerebral rhythm. No evidence has yet been obtained (Newell *et al.*, 1947b) of toxicity in man as shown by tracings of cerebral cortical electrical rhythms with the electro-encephalogram, which in the dog are affected markedly (Newell *et al.*, 1947a).

5. Factors Affecting the Potency of the Primary Dietary Hysteriagenic Agent

Apart from the amount of primary dietary hysteriagenic agent ingested there are a number of other factors which may affect the number and frequency of motor episodes very markedly. They have no primary hysteriagenic potency, but they can act as important hysteria-precipitating agents in animals already rendered hysteria-prone by ingestion of the primary agent. They fall into three categories, neuro-psychological, dietetic and metabolic.

(*a*) *Neuro-Psychological Factors*

In this category we may consider those loud noises and unusual situations which can so often in practice precipitate hysterical

episodes. Under my experimental arrangements the animals' cages were hosed out every day with a definite routine. Animals in sub-clinical stages of hysteria-proneness would cringe away from the hose instead of drinking from it, and later the hose would actually precipitate severe motor episodes. On other occasions the mere switching off and on of the lighting would cause cessation and renewal of running barking seizures, while an emotional rebuff, when an animal in a state of hysteria-proneness approached one for sympathetic caressing, would on occasion be sufficient to precipitate an episode.

There is clinical evidence that nervous stimulation arising from the alimentary tract due to constipation, ascariasis and distension by foreign bodies, may act as hysteria-precipitating stimuli, but in my experimental animals a very strict watch was kept on these factors, and constipation and ascariasis were not important in these studies.

(*b*) *Secondary Dietary Factors*

On several occasions a change of diet from one of low primary hysteriagenic potency to one devoid of any potency, *e.g.*, a pure carbohydrate diet, precipitated a series of severe motor episodes for several days. The precise mechanism of this is unknown but it is interesting to draw analogies between this observation and recent writings from the Mayo Clinic showing how effective a high fat–low carbohydrate diet can be in controlling the motor episodes of epilepsy in man (Keith, 1947). Let it suffice to draw attention to these secondary dietetic factors about which in the dog we know so little, for the carbohydrate/fat calorie ratio is probably an aspect of the problem which must not be overlooked.

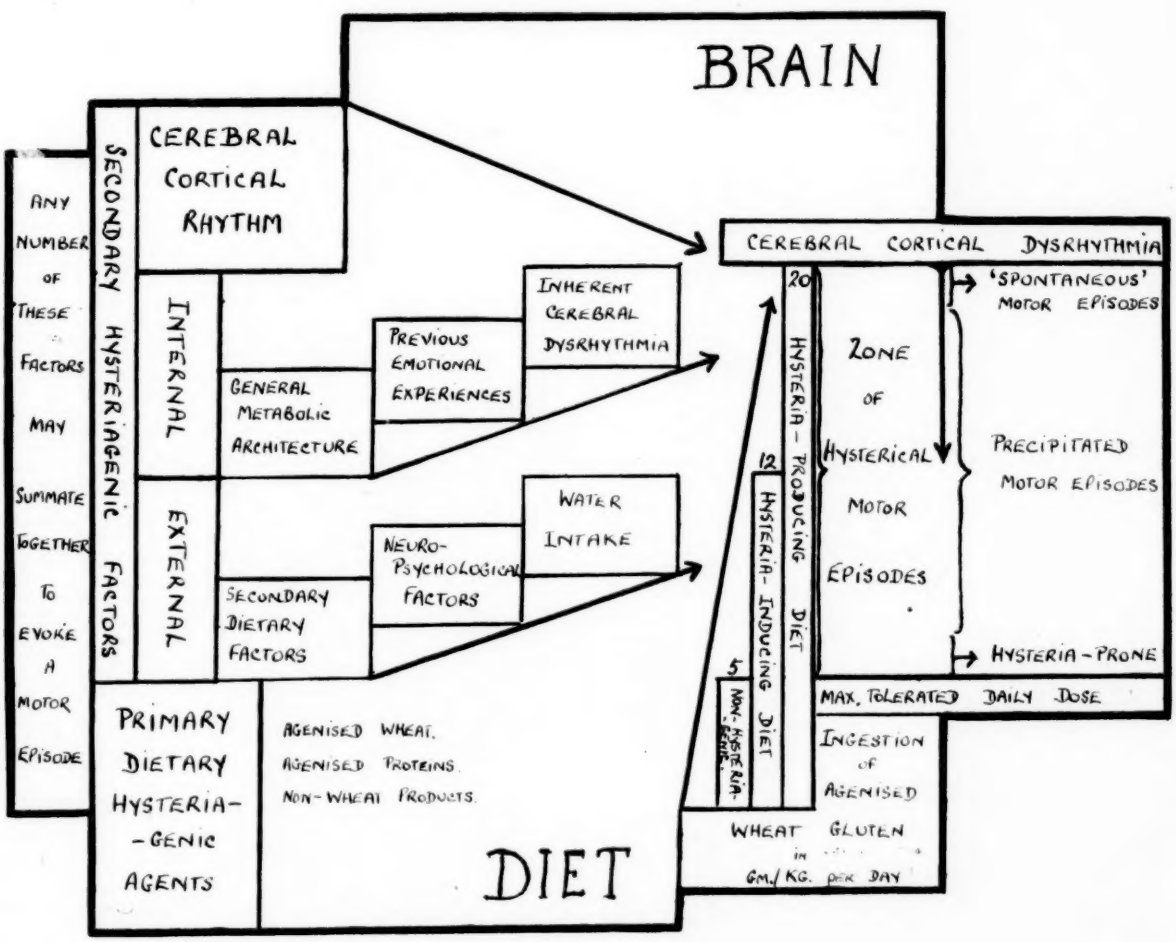
(*c*) *Metabolic Factors*

These concern the internal bodily environment as it affects the brain, in particular firstly the water and electrolyte balance, and secondly, the body saturation with certain vitamins. In man it is well known that in certain epilepsy-prone subjects the motor episodes can be precipitated by inducing water retention, and, more particularly, by increasing the water content of the brain cells. That this may apply to the dog seems likely from the observations of Rowntree (1926) on water intoxication in the dog, and Allen (1945) has stated recently that he can precipitate hysterical motor episodes in the dog by inducing severe water retention. Secondly, in an attempt to test the alleged curative effect of thiamine therapeutically, *i.e.*, in large doses over and above the normal nutritionally adequate amount given by mouth, I was able to enhance the frequency and severity of the motor episodes. In addition there are hints that exercise, and the state of the sexual cycle, also have a bearing on the problem.

The interplay of these factors is illustrated in Fig. 1.

Fig. 1.

Factors Influencing the Occurrence of Hysterical Episodes.



A diagram to illustrate the ways in which the various hysteriagenic factors can combine and summate to evoke a motor episode.

III Some Applications to Clinical Experience

With such a large number of factors involved in the actual "setting-off" of the nervous dysfunction which is expressed clinically as an hysterical motor episode, there is every opportunity for the protean manifestation of the malady as seen in practice.

The primary requirement would appear to be the establishment of a state of hysteria-proneness in the dog's brain by the ingestion of some primary hysteriagenic substance. The ease with which this state of hysteria-proneness (which may be entirely aclinical) is set up, no doubt depends in part on the inherent individuality of the dog, although any marked hereditary predisposition has yet to be demonstrated adequately. If lightly agenised wheat flour be the agent, a dog of 30 lb. will require at least 2 lb. of bread per day. The amount will vary with the degree of agenising to which the wheat flour has been subjected. Similarly, dog biscuits containing agenised flour may act as the primary hysteriagenic agent.

The actual occurrence of motor episodes, however, may never take place unless secondary dietary factors or certain precipitating factors combine to "set-off" the hysteria-prone state. Diets high in carbohydrate and low in fat and salts are more likely to exacerbate the condition, while low carbohydrate–high fat–high salt diets depress hysteria-proneness. Precipitating factors, upon whose presence the actual occurrence of motor episodes may depend, are numerous. Suitable stimulation of the visual and auditory receptors, such as the bark of another hysterical dog, a sudden noise such as a motor car engine back-firing, the noise of water from a hose, are well known to precipitate attacks. If the hysteria-prone dog be not exposed to these stimuli, it may never exhibit motor episodes. Apart from these receptors, stimulation of the nerve endings of the integument, *e.g.*, the external auditory meatus by grass awns or acari, or of the alimentary tract by foreign bodies, over-distension by large food masses or by ascariasis may serve in the hysterical-prone animal to precipitate the motor episode. In other cases certain emotional situations or unusual excitement may act as the precipitating agents. In some gun-dogs prolonged exercise may be the critical precipitating factor. In practice it is sound therapy to minimise, as far as possible, all these subsidiary factors as well as depressing the proneness of the central nervous system by sedative drugs.

Summary

The production of dietary canine hysteria depends upon a number of dietary factors, which can conveniently be placed in two categories, primary and secondary. Primary hysteriagenic factors are those which set up within the animal's central nervous system a state of hysteria-proneness, from which state the hysterical motor episodes arise. Such a primary agent is wheat protein which has been subjected to the agene process, although it is almost certain that there are other primary agents not associated with the agene process which have yet to be defined. The secondary hysteriagenic factors are those which of themselves have no power to initiate the hysterical state, but which enhance the action of the primary hysteriagenic agents; these include the carbohydrate–fat ratio, salt and factors which may influence water retention.

Diets can be placed in at least four groups according to their hysteriagenic activity:

1. *Hysteria-producing diets* are those which *produce* a state of hysteria-proneness sufficiently severe to cause "spontaneous" motor episodes.

2. *Hysteria-inducing diets* are those which *induce* a state of hysteria-proneness from which motor episodes do not occur "spontaneously" but from which motor episodes may be elicited by appropriate neuropsychological stimuli.

3. *Hysteric-precipitating diets* are those with no primary hysteriagenic potency but which, when first fed to an animal previously rendered hysteria-prone, may precipitate motor episodes.

4. *Hysteria-depressing diets* are those which reduce the liability of an hysteria-prone animal to motor episodes; one way in which they may act is by reducing the degree of water retention by the neurones of the central nervous system and by modifying in other ways the metabolic architecture of those neurones.

While the work of Mellanby (1946, 1947) and Moran (1947) has demonstrated the important role of agenised proteins as primary hysteriagenic agents, it would be unwise to assume that they are necessarily the only primary dietary factor involved in many cases of the naturally occurring disease. Furthermore, to attempt to draw from the results obtained in the dog conclusions valid for human medicine, is difficult, since we know that a maximum tolerated daily dose of an agenised protein must be ingested before any clinical disorder can occur in the dog, and that the dog has a peculiarly low M.T.D.D. compared with other laboratory animals, including the monkey. For agenised flour to be of similar importance in human medicine it would be necessary for man to show a special susceptibility similar to that of the dog, and the available evidence (Newell *et al.*, 1947b) as yet provides no support that this is so.

References

Allen, F. F. (1945.) *Amer. J. Psychiat.* **102**. 67.
Anon. (1735.) The Sportsman's Dictionary or the Country Gentlemen's Companion. Vol. 1.
Arnold, A., & Elvehjem, C. A. (1939.) *J. Amer. vet. med. Ass.* **95**. 303.
Baashuus-Jensen, J. (1935.) *Vet. J.* **91**. 339 and 379.
Bentley, H. R., *et al.* (1948.) *Nature.* **161**. 126.
Forsyth, A. A. (1947.) Personal Communication.
Grace, W. E. (1930.) *Vet. Med.* **25**. 102.
Hewetson, H. R. (1936.) *Vet. Rec.* **48**. 1202.
Hill, J. Woodroffe. (1892.) "The Management and Diseases of the Dog." 4th ed. London. Awan, Sonnenschein. pp. xii and 437.
Huxley, L. (1931.) Elizabeth Barrett Browning: Letters to her sister, 1846-59. London: Murray, pp. 30-31.
Keith, H. M. (1947.) *Proc. Staff. Meet. Mayo Clinic.* **22**. 14-16
Mellanby, E. (1946.) *Brit. med. J.* **2**. 885.
——. (1947.) *Ibid.* **2**. 288.
Melnick, D., & Cowgill, G. R. (1937.) *J. Nutrit.* **14**. 401.
Moore, James. (1863.) The Disease of Dogs, and their Homoeopathic Treatment. London: Simpkin, Marshall & Co. pp. xi to 320.
Moran, T. (1947.) *Lancet.* **253**. 289.
Newell, G. W., Erickson, T. C., Gibson, W. E., & Elvehjem, C. A. (1947a.) *Proc. soc. exp. Biol. Med.* **65**. 115.
——, ——, ——, Gershoff, S. N., & Elvehjem, C. A. (1947b.) *J. Amer. med. Assoc.* **135**. 760.
Parry, H. B. (1942.) Unpublished observations.
——. (1948.) *Lancet.* **254**. 32.
Rowntree, L. G. (1926.) *J. Pharm. exp. Therap.* **29**. 136.
Saramito, P. (1946.) Thesis. University of Toulouse.
Wagner, J. R., & Elvehjem, C. A. (1944.) *J. Nutrit.* **28**. 431.

Treatment Experiments with Trypanosoma vivax (Ziemann) Disease of Cattle

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The significance of the part played by *Trypanosoma vivax* in the causation of trypanosomiasis in Africa has been the subject of some discussion and it is plain that the views of experienced workers are at variance. Contributors to correspondence in *The Veterinary Record* [vide (1945) p. 585; (1946) p. 80, p. 178 and p. 200] have, however, all failed to notice the report of Hudson (1944) in which he described experiments with a strain of the parasite, which caused a heavy mortality amongst cattle following a short septicaemic course of the disease.

Other reports of trypanosomiasis outbreaks due to *T. vivax* are received from time to time from various localities in Kenya, in which the parasite has behaved similarly, causing a heavy death-rate.

Hudson (1944) compared such outbreaks in cattle with those of *T. simiae* in pigs, where trypanosomes are present in the blood in enormous numbers. It is, therefore, evident that as with other trypanosomes, *T. vivax* shows all stages of virulence between a fulminating type and one that is certainly benign, if not almost non-pathogenic.

Its importance, however, in the pathogenesis of trypanosomiasis in cattle is not confined to the fulminating strain, since experience shows that cattle, apparently normal but harbouring the trypanosome, often succumb to fatal trypanosomiasis if subjected to adverse conditions, such as ill-health, overwork, or even the injection of prophylactic agents, particularly attenuated rinderpest goat virus.

Successful treatment of infected herds is thus a matter of some importance and the criterion of success should be the total elimination of the parasites, since otherwise there may be a subsequent heavy mortality under the stress of adverse conditions.

Experimental work with this parasite presents great difficulty. The vast majority of strains are benign. Following a period of two to three weeks, during which parasites are regularly present in the blood, the host animal starts on the path to recovery. Parasites disappear from the blood and can only be found there at irregular intervals, sometimes only one to two days per month. The investigator is, therefore, at a loss to know whether recovery is due to his drugs or whether it is occurring in the natural course of events.

In the work described here, Hudson's fulminating strain of the parasite has been used to test various drugs. This was maintained in cattle by Dr. E. Aneurin Lewis by needle passage. By this means, its former virulence was lost and it resumed the attributes of a benign strain of the parasite. On transmission through *Glossina*, however, it regained its former virulent properties. I am indebted to Dr. Lewis for infecting cattle for me with this strain, by means of tsetse transmission.

With this strain, death normally occurred in 15 to 21 days following an acute critical period. If this period was passed, the animal died some few weeks later. When no treatment was given the parasite invariably proved fatal.

By use of this strain of *T. vivax*, therefore, difficulties of investigation were overcome and the effects of drugs on blood forms of the parasite could be tested. The drugs tested were Bayer 7602, phenanthridinium 897 and stibophen. The Bayer 7602 was supplied to us free of charge by Imperial Chemical (Pharmaceuticals), Ltd., under the label of M. 3024. Its trial followed reports of successful treatment of *T. vivax* by Kubes (1944) in Venezuela. Dosage is given as grammes (g.) per 100 kg. body weight, given intravenously.

Treatment with Bayer 7602

The Benign Disease.—Two cattle, Nos. X80 and 9893, were infected by needle transmission on 21.1.46. Parasites appeared in the blood on 4.2.46 and 6.2.46 respectively. Both animals received Bayer 7602 on 14.2.46, at 1 g. per 100 kg. On 15.2.46, trypanosomes disappeared from the blood, but reappeared in the case of X80 on 16.2.46, and both cattle had positive blood slides on 19.2.46. Both subsequently died of chronic trypanosomiasis.

The Acute Disease.—Two animals, Nos. 567 and 556, were infected on 5.1.47 with *T. vivax* by means of *Glossina austeni*. Both animals suffered from fulminating *T. vivax* infection and were treated on 16.1.47. No. 556 received 2 g. per 100 kg. and died from its effects within two minutes. No. 567 received 1 g. per 100 kg.; this had no effect on the course of the disease and the animal died of acute trypanosomiasis three days later.

In view of these results it was not considered worth while to pursue the investigation further.

Treatment with Phenanthridinium 897

Six animals suffering from fulminating *T. vivax* infection, were treated with phenanthridinium 897 at 2 mg. per 100 kg. of body weight. One animal suffered a relapse four weeks after treatment and died of acute trypanosomiasis. The other five made an eventual recovery.

Treatment with Stibophen

Normally, four weekly injections of stibophen are given in treatment of trypanosomiasis. In this case, only one injection was used at the usual dosage level, that is by the use of one tablet of 0·5 g. Six animals in all were treated, of which two died within a week from trypanosomiasis. Of the four remaining animals two suffered again from acute trypanosomiasis and were treated with phenanthridinium 897 and recovered. The other two animals recovered.

Discussion

There is no evidence that Bayer 7602 has any effect on the course of *T. vivax* disease either in its chronic or acute types. It may be said in this connection that the strain of parasite used has been proved by Dr. Lewis to be a true *T. vivax* by careful measurement and that it has only one size range.

With both stibophen and phenanthridinium an unusual feature has been that the parasite has not been eliminated by the treatment. In every case, animals that recovered have shown trypanosomes in the blood sooner or later following treatment. Neither drug, therefore, has cured; it has merely diverted the disease from a fatal to a benign course. Neither can, therefore, be regarded as fully effective.

Of the two drugs plainly phenanthridinium is more effective than stibophen and must be regarded as the drug of choice in acute cases of this trypanosomiasis. Stibophen also has a certain value which might be enhanced by giving the usual course of four injections; the two deaths within a week of treatment, however, show that its value is less than that of phenanthridinium.

While stibophen and phenanthridinium are both effective to a degree in the acute trypanosomiasis, it remains doubtful whether they can be effective in eliminating the "carrier" animal which may succumb under adverse conditions. The recovery course as indicated by temperature curves and weekly red cell counts, was slow and it is clear that these drugs may have only a slight effect on the chronic type of disease. It is of great interest that they should have converted a virulent strain of the trypanosome into a benign one.

References

Hudson J. R. (1944.) *J. comp. Path.* **54**. 108–119.

Kubes, Vladimir. (1944.) 3a Conf. interameric. Agric. Caracas No. 1—English summary, pages 111–117.

Weekly Wisdom

It was as important to the country that the young should chance their arm at 18, *as that the old should be sure of their pensions at* 65. *A boy who entered a career at* 18 *because he was assured of a pension* 45 *years on was looking at life backwards.*—Mr. R. A. Butler.

CLINICAL COMMUNICATION

"Double" Intussusception of the Small Intestine in the Cow

ROBERT A. BROWN, M.R.C.V.S.

Paulton, Somerset

Subject.—Fourteen-year-old Shorthorn-cross cow at pasture.

History.—The cow had been found to be very uneasy at the afternoon milking on May 27th. She had got up and down frequently and kicked at her abdomen. She gave practically no milk and had given her normal amount at morning milking.

I was called to see the animal the following day. She had been kept in the stall during the night and had passed some faeces, normal in appearance according to the owner. Cut grass had been offered to her but she had not eaten.

Examination.—General appearance quite bright. Occasionally she kicked at her abdomen. Temperature, 101·4°. Pulse, accelerated. Complete absence of ruminal and bowel sounds. The rumen was well filled and "doughy" on palpation in the flank. Deep abdominal palpation on both sides produced no evidence of pain, nor did the usual tests for traumatic gastritis.

No diagnosis was made at this stage. A mixture consisting of the usual gastric stimulants was prescribed, together with draughts of black treacle.

Next day temperature was 99°. No faeces had been passed and she had not eaten. She was by no means moribund in spite of subnormal temperature. She frequently adopted the position of placing her fore feet as far forward and her hind feet as far back as possible. She still kicked at her abdomen.

Rectal examination caused much discomfort and straining. The rectal wall clung tightly to the arm. On withdrawal, the arm was coated with foetid mucus and tarry, altered blood.

Diagnosis.—A diagnosis of intussusception, probably of the small intestine, was now made. The straining made it quite impossible to attempt palpation of the intussusception per rectum.

I gave a grave prognosis, while pointing out the possibility of sloughing taking place. Next morning, however, the owner requested that she be slaughtered.

Post-mortem Examination.—The entire length of the small intestine was tympanitic. The intussusception was not at first obvious, there being no inflammatory changes on the outer serous surface. It was situated about 2 feet in front of the ileo-caecal valve, and was itself about 15 inches in length.

Reduction revealed that it was in fact a double intussusception. Apparently one invagination had taken place, and this in turn had invaginated into the distal part of the intestine. The invaginated parts were in an advanced state of inflammatory change.

Discussion.—The case is considered worth recording because of:

(*a*) The amazingly bright appearance of the animal throughout.

(*b*) The absence of any febrile reaction.

(*c*) The double nature of the intussusception.

I should be glad to hear from any practitioner who has had personal xperience of successful sloughing taking place.

REVIEW

["**Sheep Production.**" By Allan Fraser. (Nelson, 7s. 6d.)]

No one could deny, for instance, when reading Kipling, that he knew India; but always there is the query at the back of one's mind—did he know Indians? Or whether Wells, so admirable at Outlines, knew anything of History. No such doubts can assail the reader of Allan Fraser's "Sheep Production"; one feels from the first chapter or so—"this man *knows* sheep." He begins with a review, geographical and historical, of the sheep industry, stressing that among the primitive pastoral people, the sheep was a triple-purpose animal—meat, wool and milk. The emphasis gradually shifted to wool and then to meat. A description of breeds follows, passing on to discussions of methods of breeding and selection for various purposes and environments. Much sound advice is given on feeding and management, with the encouraging thesis (in contradistinction to figures recently published by the Agricultural Economics Branch of Liverpool University!) that sheep-farming may yet be profitable. This excellent study closes with some observations and information on mutton and wool, their classes and uses. The final chapter on sheep diseases, while simple and clear, does not enter into sufficient detail to be of interest to veterinarians. But the book is heartily to be recommended to all members of the profession interested in agronomy and the wider background of their day-to-day activities

Questions and Answers

The submission of questions for inclusion in this column will be welcomed: they can relate to any aspect of veterinary work. For purposes of record, each question is numbered, and those submitting questions are required to furnish (not for publication) name and address.

Answers to readers' queries represent exclusively the personal opinions of the writers, and criticism of the replies will be appreciated.

All communications should be addressed to the Editor.

Treatment of Colic

Q. CCLI.—*Is there a general treatment for colic which may be considered to have surpassed Reek's treatment? Do you recommend the intravenous treatment of barium chloride?*

A.—Caulton Reeks wrote: "There is no reliable rule of thumb in medicine. Each case must be treated on its own merits." He would surely have resented the suggestion that he had a single remedy for colic.

He advised that sedative treatment was liable to cloak rather than to cure the condition and he preferred stimulants and laxatives of which he discussed a great variety. He recommended, in many cases, the treatment so strongly advocated by W. W. Lang—large quantities of salt and water by the stomach tube with other drugs added. The writer finds that a great many of his cases respond to this hypertonic saline but he is of the opinion that the choice of drugs in the treatment of colic is very largely an affair of geography. Colics in one district may differ from those in another where the type of horse and the work and feeding are different.

Barium chloride, acetyl choline and arecoline have their enthusiastic adherents but these drugs have the disadvantages of being only transient in their effects and of being dangerous if used on unsuitable cases. There seems to be no reason to differ from Reeks on the subject of barium chloride. He wrote, quoting from Quitman: "When barium chloride acts, it acts beautifully, when it kills, it kills quickly and it does the latter frequently. It is intensely painful in its effect and can bring in the end only shame and discredit on the user."

. . . . .

Transport of Dangerous Bulls

Q. CCLII.—*When dangerous bulls are to be consigned by rail or motor vehicle to a slaughterhouse, the query often arises as to whether some narcotic could be given to the animal to render it more tractable during the journey. Unfortunately, many drugs administered shortly before slaughter cause an abnormal taste or odour of the meat, and this may be so marked as to render the carcase unmarketable. It is stated chloral hydrate and bromides are drugs which have this effect, and it would be interesting to know how narcosis may be induced in bulls without deteriorating the value of the carcase or rendering it unfit for food.*

A.—The writer knows of no narcotic or sedative which will render dangerous bulls tractable without the liability to meat taint and, in our present state of knowledge, he would be reluctant to advise the use of such agents prior to a journey.

A bull, if deeply narcotised, might well roll on its side during transit, become blown and die from asphyxia. If a sub-clinical dose were given it might produce excitability. A bull, recovering from an effective narcotic or sedative, might become excited and more dangerous than normally, whilst the fact that the bull had been "doped" might lead the attendants to neglect to maintain the necessary caution. It would be very difficult to estimate the correct dose necessary, having regard to the varying lengths of journeys by road or rail with, perhaps, unforeseen delays.

In man, the anti-histaminic substances are known to produce drowsiness and if, as a result of the use of such agents in veterinary practice (*Vet. Rec.* **60**. 257), this subsidiary action is noted in animals, the use of such a substance might prove useful, for these agents are rapidly excreted from the body and a short interval of rest at the slaughterhouse would be sufficient to ensure freedom of the body from such an agent even if it were liable to give rise to meat taint. Another possibility is the use of a quick acting barbiturate, such as hexabarbitone which also is rapidly excreted.

The writer knows of a dealer who, for over 20 years, made a speciality of buying, transporting and slaughtering intractable and dangerous bulls. His method was to put "blinds" on the bull. A rope was then fastened around the horns or poll and passed through the bull ring. The bull was then led with a "bull leading pole" in the ring; occasionally two men with poles were employed, one on each side of the animal. The bull was halted at the entrance to the truck whilst the end of the rope was passed through a ring or other part of the conveyance. The leading pole was detached and the bull loaded into the vehicle by a steady pull on the rope.

Masking will almost invariably quieten intractable animals. The respondent knows of a vicious bull which was blind-folded and led to the slaughterhouse, for a distance of three miles, through the busy streets of the East End of London—personally, he would hesitate to advise this as a regular procedure.

Poisoning from the Use of Slug Killers Containing Metaldehyde

Q. CCLIII.—*I have recently had the misfortune of meeting two fatal cases of meta (slug poison) poisoning in corgi dogs. Could you please tell me if there is any effective method of treatment?*

A.—The above question calls to mind the danger of poisoning to small animals due to the increasingly widespread use of slug killers containing metaldehyde as their active principle. It seems therefore appropriate to review briefly the whole subject.

Toxicity.—Pure metaldehyde, "Meta" solid fuel, is known to have caused the deaths of small animals and young children.

Proprietary slug killers having "meta" as their active principle have also caused deaths in dogs, particularly young puppies, attracted by the base in which the "Meta" is mixed. As examples of toxicity, one tablet (approximately 3 grammes) "Meta" killed a two and a half-year-old girl and a 4 kg. dog showed signs of poisoning after eating half a tablet (approximately 1·5 grammes).

Commercial slug killers contain approximately 3·5 per cent. "Meta" (*i.e.*, for a 1·5 gramme dose about 1½ oz. of slug killer would have to be eaten).

Mode of Action and Symptoms.—Metaldehyde is a narcotic poison and the symptoms are incoordination, tendency to circle, gross increase in respiratory rate, loss of consciousness, narcotic excitement (paddling of limbs, etc.) and cyanosis of membranes. Pulse is rapid and bounding. Temperature is depressed.

Death occurs from respiratory failure.

If the narcotic stage is survived there is a slow lightening of narcosis over about 12 to 24 hours in a moderately severe case. Death may occur in three to four days from secondary gross liver damage (cf., chloroform poisoning).

Treatment.—There is no specific antidote. Treatment consists in

(1) The prevention of absorption by prompt vomiting. The owner may be instructed to give washing soda if the history is definite and narcosis is not yet too deep, or apomorphine 1/30th to 1/10th gr. subcutaneously may be given.

(2) Measures taken to combat symptoms:—

(*a*) The use of cardiac and respiratory stimulants, *e.g.*, nikethamide, adrenalin, etc., by injection. Inhalation of CO_2-O_2 mixture and the application where necessary of artificial respiration.

(*b*) Prevention of loss of body warmth by blankets, hot bottles, etc.

(3) Measures taken to assist detoxication and bolster the liver, *e.g.*, injection of glucose saline and/or calcium dextrogluconate.

Prognosis.—This depends upon the amount ingested and the rapidity of absorption from a full or empty stomach.

Where narcosis goes beyond medium depth prognosis is always grave.

* * * * *

Correspondence

A Uterine Catheter Difficulty

Sir,—Surely the most straightforward method of passing the uterine catheter in the cow (not heifer) must be with the aid of Vulsellum forceps, the use of which enables one to grasp the cervix directly, without fumbling with it through the rectum.

The teeth of the forceps are guided by the left hand into the vagina, and clipped firmly into the lower lip of cervix. The cervix is retracted by means of traction, both with the forceps, by the right hand, and directly by the fingers of the left hand.

When retracted as far as is safely possible, the cervix can be firmly grasped with the whole hand. The operation is completed by urging the catheter forwards through the os, and at the same time helping it through the maze of folds by easing the cervix over the catheter.

Cases are occasionally met with where it seems impossible to pass the catheter. It is better to "give in" after a few minutes than cause an acute traumatic cervicitis, which is exactly the type of ailment one is treating.

Yours faithfully,

R. D. Franklin.

31, Cleveland Road,
Wolverhampton.
July 18th, 1948.

At the anniversary meeting of the Linnean Society of London held on May 24th, the Linnean Gold Medal was presented to Dr. Agnes Arber, and Trail Awards and Medals were presented to Dr. Honor Bridget Fell and Dr. Cyril Dean Darlington. The following were elected officers for the session 1948-49: President: Professor G. R. de Beer; Treasurer: Colonel F. C. Stern; Secretaries: Dr. B. Barnes (Botany), Dr. A. Tindell Hopwood (Zoology). The new members of Council were Mr. James M. Fisher, Mr. N. B. Kinnear, Professor W. H. Pearsall, Dr. W. R. Philipson and Mr. N. Y. Sandwith.

QUESTIONS IN PARLIAMENT

Prior to the rising of the House of Commons for the Summer Recess, the questions answered included the following:—

* * * * *

Bovine Mastitis

Mr. J. Lewis asked the Minister of Agriculture how many millions of gallons of milk it is estimated are lost every year due to the incidence of bovine mastitis.

Mr. T. Williams: Mastitis is not a notifiable disease, and I am afraid that I cannot give any reliable estimate of the loss of milk caused by it.

Mr. J. Lewis asked the Minister of Agriculture what experiments have been carried out by his Department in the treatment of bovine mastitis by means of penicillin; what results have been achieved; and what quantity of penicillin it is estimated would be required in order for it to be effective in combating this disease.

Mr. T. Williams: As a result of the research work carried out in the laboratory and in the field since penicillin became available for veterinary purposes, it has come into general use for the treatment of cows infected with streptococcal mastitis with generally satisfactory results. Research is now proceeding into more intensive methods of eradicating the disease from infected herds. I regret I cannot give any estimate of the total quantity of penicillin that would be required to deal with streptococcal mastitis in our herds, but to treat one infected cow between one quarter and two mega units of penicillin are required, according to circumstances.

* * * * *

Tuberculin-Tested Milk

Mr. Piratin asked the Minister of Food to what extent T.T. milk not bottled by the farmer is subsequently kept separate by the bottles and sold as T.T.; and what steps he takes to ensure that such milk is not mixed with non-T.T. milk.

Dr. Summerskill: In May, 1948, the latest month for which returns are available, approximately 27 per cent. of the T.T. milk sold by producer-wholesalers in England and Wales was retailed as such. Under the Milk (Special Designations) Regulations, T.T. milk sold as such must be kept separate from all other milk and sold to the consumer in sealed containers specially labelled and fitted with overlapping caps. Failure to comply with these conditions may involve dairymen in the revocation or suspension of their licences to sell T.T. milk.

Mr. Piratin: While thanking the Minister for that information, may I ask her whether it is not the case that a certain amount of T.T. milk is not sold as T.T. milk at all and is, therefore, wasted so far as the quality is concerned, since it is mixed with ordinary milk before being sold? The purpose of this Question is to try to get the matter put right. T.T. milk should be sold as such, because there is a wide demand for it.

Dr. Summerskill: Unfortunately, the hon. Gentleman is wrong. I wish there were a wide demand for it. Only 27 per cent. is sold as T.T. milk. We cannot govern consumer preference.

Mr. E. P. Smith: Does not the hon. Lady agree that the only way to ensure that there is pure milk is to have it boiled in every household?

Hon. Members: No.

* * * * *

Poultry Exhibitions (Restrictions)

Mr. Frederic Harris asked the Minister of Agriculture when it is proposed to lift the ban on the showing of poultry in public exhibitions.

Mr. T. Williams: I fully realise the desirability of relaxing some of the restrictions that have had to be imposed on account of fowl pest as soon as it is reasonably safe to do so. The position is being watched all the time; but I am afraid I cannot make any further announcement at the moment.

UNIVERSITY OF WALES AND A VETERINARY FACULTY

Although Wales is not mentioned in the Government Veterinary Surgeons Act, the Welsh University is pressing its case for the establishment of a Faculty to teach veterinary science.

The *Liverpool Daily Post* reports that at a meeting of the University Court it was decided to prepare a scheme and to approach the University Grants Committee for financial assistance. It has been suggested that the proposed institution for veterinary education should not be identified with any existing department of the university, but should be autonomous in finance and administration. The provision of full chairs is contemplated, and the opinion has been expressed that to accept a second best or makeshift arrangement, merely in order to make a start, would be fatal to the success of the school.

NOTES AND NEWS

Diary of Events

Aug. 12th.—Summer Meeting of the Eastern Counties Division, N.V.M.A., at Cromer. (*See Notice.*)

Aug. 13th.—Meeting of the Yorkshire Division, N.V.M.A., at Leeds (Hotel Metropole), 2.30 p.m.

Sept. 8th to 15th (inclusive).—N.V.M.A. Congress at Southport.

Sept. 11th.—13th Annual Meeting and Dinner of the Royal (Dick) Veterinary College, Alumnus Association at Southport (Prince of Wales Hotel), 7.30 p.m.

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The Veterinary Surgeons Act

The Veterinary Surgeons Act was among those to which the Royal Commission which sat in the House of Lords on Friday, July 30th, signified the Royal Assent.

* * * * *

R.C.V.S. Register, 1948

The Register of Veterinary Surgeons for the year 1948 has now been published and distributed to members of the Royal College. Any member failing to receive a copy should inform the Registrar at 10, Red Lion Square, London, W.C.1.

* * * * *

Children's Visit to Denmark

As readers will have noted from announcements in our columns, the Danish Veterinary Association, through its President, Dr. Plum, this year renewed its hospitable invitation to children of British veterinary surgeons to go for a holiday in Denmark.

A party of 23 left on Tuesday, August 3rd, for what promises to be a very lovely holiday in another country and the President, Mr. L. Guy Anderson, and the General Secretary, Mr. F. Knight, saw them all off at Liverpool Street, London, with Miss Rosa Smith, of the Headquarters staff, in charge of the happy group. The N.V.M.A. was very glad to play its part in arranging this trip and understands that some of the children have been in correspondence with their future hosts and hostesses, all of whom seem very happy to welcome the children in their homes. They will be staying in many different places all over the country—from well into the north of Jutland to a place far in the south on the island of Moen. Several, too, will be staying in or near Copenhagen.

* * * * *

Petrol Rationing Scheme

Members are reminded that, if they have not already done so, they should make immediate application to the local Post Office for their basic petrol ration.

* * * * *

PERSONAL

Animal Health Trust Appointment.—Colonel John Hickman, M.R.C.V.S., Reader in Surgery at the Royal Veterinary College, has been appointed to the position of Surgeon at the Equine Research Station of the Animal Health Trust at Newmarket.

* * * * *

Births.—Case.—On July 26th, at Cardiff, to Joan (formerly Farman, M.R.C.V.S.) and Robert Case—a daughter, Rowena Gillian.

Daykin.—On Thursday, July 29th, 1948, at St. Mary's, Raleigh Street, Nottingham, to Elizabeth (*née* Sales) and Philip W. Daykin, B.SC., M.R.C.V.S.—a son, John Michael.

Gooch.—On Wednesday, July 28th, 1948, at Stamford General Hospital, to Betty (*née* White), wife of F. W. L. Gooch, M.R.C.V.S., of Stamford, Lincs., a brother for Roger and Brian—Stephen Leeds.

Rawlinson.—At St. Mary's Place, Ulverston, to Nora, wife of W. E. Rawlinson, M.R.C.V.S.—a son.

* * * * *

Marriages.—MacLeod—Evans.—On July 3rd, 1948, at St. Mary's Church, Pembridge, Herefordshire, Donald MacLeod, M.R.C.V.S., eldest son of Mr. and Mrs. J. MacLeod, of Portnalong, Portree, Skye, to Gwendoline Mary Evans, S.R.N., only daughter of Mr. and Mrs. W. S. Evans, of Westcroft, Pembridge. The Rev. G. L. Bourdillon officiated and Mr. John N. Peatt, M.R.C.V.S., was best man.

Schwabacher—Lethbridge.—The marriage took place on July 30th, 1948, at Watford, between Miss G. Lethbridge and E. N. Schwabacher, M.R.C.V.S.

Shaw—Patchett.—On June 23rd, 1948, E. Hilda Patchett, B.V.SC., M.R.C.V.S., to Josiah A. Shaw, both of Chorlton-cum-Hardy, Manchester.

Captain T. L. Wright.—Our readers will be glad to learn that Captain T. L. Wright, who, as recorded in our issue of July 24th, is in St. George's Hospital after having been knocked down by a car in Piccadilly, is in excellent general health. The plating of the fracture he sustained (which was a "butterfly" and not a "compound" as was then stated) has been delayed, however, by the skin blistering caused by the original temporary plaster.

Captain Wright desires, through our columns, to express his great appreciation of the sympathy and offers of help in the numerous letters sent him by members, individual reply to which eventually has proved too big an undertaking. Among the many deprivations attendant upon the accident was the inability of Captain and Mrs. Wright to attend the Royal Garden Party on July 22nd.

* * * * *

HAMPSHIRE SOCIETY'S A.I. PROGRESS

Members of Hampshire Cattle Breeders' Society, who elected Lord Selborne as their President, were told at their annual meeting at Southampton (says *The Farmer and Stock-Breeder*) that 12,676 cows were inseminated during 1947, compared with 6,769 during the previous year. This work is still steadily increasing, for Mr. G. N. Gould, Chairman of the Committee of Management, revealed that 9,817 cows had already received first inseminations during the first six months of 1948.

Total membership of the Society at the end of 1947 was 1,623, 771 having joined during the year. Estimated numbers of cows in the herds of members was 20,161. Outside Hampshire and the Isle of Wight there are members in Sussex, Wiltshire and Berkshire.

There was a notable increase in fees during the year, and revenue from this source was £24,487—double the comparative figure for the previous year. Fees for insemination have been reduced from 40s. to 35s.

While the premises at Lyndhurst had been further adapted for the accommodation of 19 bulls during 1947, the accommodation would be insufficient for ultimate requirements. It had been decided that the most practical course would be the purchase of a suitable farm to which the centre could be moved gradually, when the time was opportune.

The only warning note in the report dealt with the supply of new cars. "It really is extraordinary," said Mr. Gould, "that a specialist service doing such work as we do can obtain no degree of priority for transport."

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the N.V.M.A.

Correspondents are requested to write as briefly as possible

JEWISH AND MOHAMMEDAN METHODS OF SLAUGHTER

Sir,—It is hardly necessary to reply to the points raised in the letter from the Editor of the *Islamic Review*, as far as your professional readers are concerned, since they will readily appreciate that, in the main, the views of the Ex-Imam of the Woking Mosque are based on false assumptions.

For the benefit of lay readers of the *Record*, however, I must state that stunning with a captive bolt pistol does not prevent efficient bleeding. The statement that "Stunning . . . has a paralysing effect on all muscles, etc.," is incorrect, since some muscular movement is actually stimulated after stunning by the captive bolt pistol. The heart beat is stronger and reflex movements occur. Animals do not die instantaneously as a result of stunning—even with the loose bullet, in the majority of cases, the heart continues to beat for some minutes. Thirty years or so ago, I was able to feel a distinct heart beat through the chest wall, with the palms of my hands, for upwards of five minutes after cavalry horses were killed with a loose bullet.

The contention that "the pain involved in killing is the same whether, when death occurs, the animal is conscious or unconscious," is scientifically illogical.

Having spent a few years in the East, I am not unacquainted with the fact that transport bullocks are cast for shoeing, but, I submit, this cannot fairly be compared with the casting and positioning for ritual slaughter.

The public attitude to the humane treatment of animals in the East is, in general, not so enlightened as in this land.

There is very little ritual slaughtering by Muslims in this country and my experience is confined to the occasional slaughter of sheep or goats by Muslim members of a ship in our ports. Unlike the Jewish ritual slaughter, Muslim ritual slaughter is carried out by a Muslim member of the crew irrespective of any qualification or experience in the work. They usually arrive at the slaughterhouse without knife or other tools which they have to borrow from the slaughtermen, and few good workmen are willing to lend their best tools to a stranger.

Yours faithfully,

County Borough of West Ham, H. E. Bywater.
Essex House,
High Street, Stratford, E.15.
July 20th, 1948.

* * * * *

The following is an extract from a letter recently received from Dr. J. H. Mason, M.R.C.V.S., of the South African Institute for Medical Research, Johannesburg:—

"I may be able to add one bit of information *re* unconsciousness and loss of blood. We occasionally bleed horses 'out' from the carotid. They receive 1½ oz. chloral hydrate intravenously, are pulled on to mattresses (real mattresses) when wobbly and then chloroform is administered. The carotid is exposed and a cannula inserted. On two occasions, after 15 litres of blood had been tapped, I removed the chloroform on the assumption that consciousness would not return, because of the loss of blood. After 25 litres had been taken (say two-thirds of the total blood volume) consciousness did return—struggling, eye reflex present. I shot the horses after I had taken 27·5 litres of blood because by that time the blood flow was slow and clotting was occurring in the cannula. But both animals responded to stimuli, and would have made attempts to rise, if the hobbles had not been *in situ*."

ADVERTISERS' ANNOUNCEMENTS

Inauguration of Newton Victor Limited

As announced in your last issue, on July 31st a new company—Newton Victor Limited—came into being, amalgamating Victor X-ray Corporation, Ltd., Newton & Wright, Ltd., and the X-ray research and manufacturing activities of Metropolitan-Vickers Electrical Company, Ltd. Through branches and service establishments located in all principal centres throughout the British Isles it will continue and expand the facilities hitherto provided by its constituent members to users and prospective users of veterinary and medical X-ray and physiotherapy apparatus. The combination of material resources, experienced personnel and productive potential represented by the amalgamation of these three organisations in Newton Victor Limited is of particular significance at the present time, when the widening fields of medical and veterinary radiology imply greater demands upon the British X-ray and electromedical industry.

Head offices of Newton Victor Limited will be at 15, Cavendish Place, London, W.1. Branch offices and service establishments at Belfast, Birmingham, Bristol, Cambridge, Cardiff, Dublin, Exeter, Glasgow, Leeds, Liverpool, Manchester, Newcastle-upon-Tyne, Nottingham, Oxford, Sheffield, Southampton.

DISEASES OF ANIMALS ACTS, 1894 TO 1937, AND AGRICULTURE ACT, 1937 (PART IV).

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period	Anthrax	Foot-and-mouth	Fowl Pest	Parasitic Mange*	Sheep Scab	Swine Fever
July 1st to 15th, 1948	3	—	5	—	1	1
Corresponding period in—						
1947	1	3	47	—	9	—
1946	6	1	—	—	2	7
1945	5	13	—	—	6	34
Jan. 1st to July 15th, '48	50	—	211	1	27	21
Corresponding period in—						
1947	48	101	589	1	34	16
1946	57	29	—	4	38	282
1945	59	98	—	5	46	542

NOTE.—The figures for the current year are approximate only.

* Excluding outbreaks in Army Horses.

Tuberculosis Order of 1938

The number of bovine animals slaughtered under the Tuberculosis Order of 1938 during the period April 1st to June 30th, 1948, was 1,739.